

10/8/4, 195
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(FILE 'HOME' ENTERED AT 12:33:47 ON 10 MAY 2005)

FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, CANCERLIT' ENTERED AT 12:34:08 ON
10 MAY 2005

L1 0 S PHOSPHOCOLINE? AND PHOSPHORYCHOLINE?
L2 38 S PHOSPHOCOLINE?
L3 117 S PHOSPHORYCHOLINE?
L4 139290 S PHOSPHATIDYLCHOLINE?
L5 0 S LYSOPHOSHAYIDYLCHOLINE
L6 19895 S LYSOPHOSPHATIDYLCHOLINE?
L7 0 S L2 AND ANTIBOD?
L8 38 S L3 AND ANTIBOD?
L9 38 S L3 AND ANTIBOD?
L10 4651 S L4 AND ANTIBOD?
L11 675 S L6 AND ANTIBOD?
L12 31 DUPLICATE REMOVE L8 (7 DUPLICATES REMOVED)
L13 31 DUPLICATE REMOVE L9 (7 DUPLICATES REMOVED)
L14 2390 DUPLICATE REMOVE L10 (2261 DUPLICATES REMOVED)
L15 340 DUPLICATE REMOVE L11 (335 DUPLICATES REMOVED)
L16 0 S L13 AND PAF?
L17 0 S L12 AND KIT?
L18 31 S L12 AND L13
L19 1 S L18 AND CARDIO?
L20 1 S L18 AND ATHEROSCLEROSIS?
L21 0 S L3 AND ARTEROSCLEROSIS?
L22 1 S L3 AND L6
L23 465 S L4 AND HYPERTENSION?
L24 94 S L6 AND HYPERTENSION?
L25 0 S L23 AND BLODD?
L26 259 S L23 AND BLOOD?
L27 50 S L24 AND BLOOD?
L28 202 DUPLICATE REMOVE L26 (57 DUPLICATES REMOVED)
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L30 3 S L28 AND KIT?

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FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, CANCERLIT, JAPIO' ENTERED AT
15:12:40 ON 10 MAY 2005

L1 44219 S (PLATELET ACTIVATING FACTOR)
L2 0 S L1 AND ATHROSCLER?
L3 807 S L1 AND ATHEROSC?
L4 1245 S L1 AND PHOSPHORYLCHOLINE?
L5 1245 S L1 AND PHOSPHORYLCHOLINE?
L6 2675 S L1 AND PHOSPHOCHOLINE?
L7 130 S L5 AND L6
L8 1425 S L1 AND PHOSPHATIDYLCHOLINE?
L9 908 S L1 AND LYSOPHOSPHATIDYLCHOLINE?
L10 262 S L8 AND L9
L11 2 S L10 AND L7
L12 2 DUPLICATE REMOVE L11 (0 DUPLICATES REMOVED)
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L14 0 S L10 AND REVIEW?
L15 29 S L10 AND ASSAY?
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L18 97 S L3 AND L9
L19 101 S L4 AND L8
L20 106 S L4 AND L9
L21 14 S L19 AND L6
L22 10 S L20 AND L6
L23 14 DUPLICATE REMOVE L21 (0 DUPLICATES REMOVED)
L24 10 DUPLICATE REMOVE L22 (0 DUPLICATES REMOVED)
L25 2 S L23 AND L24

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on STN

AN 1998195672 EMBASE

TI Metabolic fate of **platelet-activating factor**

(PAF, 1-O-alkyl-2-acetyl- sn-glycero-3-phosphocholine) and lyso-PAF (1-O-alkyl-2-lyso-sn-glycero-3- phosphocholine) in FRTL5 cells.

AU Botitsi E.; Mavri-Vavayanni M.; Siafaka-Kapadai A.

CS A. Siafaka-Kapadai, Dept. of Chemistry (Biochemistry), University of Athens, Zografou, 15771 Athens, Greece

SO Journal of Lipid Research, (1998) Vol. 39, No. 6, pp. 1295-1304.

Refs: 47

ISSN: 0022-2275 CODEN: JLPRAW

CY United States

DT Journal; Article

FS 029 Clinical Biochemistry

LA English

SL English

ED Entered STN: 19980716

Last Updated on STN: 19980716

AB The metabolism of **platelet-activating factor**

(PAF, 1-O-alkyl-2-acetyl- sn-glycero-3-phosphocholine) and lyso-PAF (1-O-alkyl-2-lyso-sn-glycero-3- phosphocholine) was investigated in FRTL5 cells, a normal rat thyroid cell line. FRTL5 cells incorporated [3H]PAF and deacetylated this compound to the corresponding [3H]lyso-PAF which was not accumulated or secreted but converted mainly to alkyl-acyl-phosphocholine indicating that this acylation process was particularly active in these cells. Among metabolic products of both [3H]PAF and [3H]lyso-PAF were alkylglycerol as well as its mono- and diacyl derivatives. [3H]alkylglycerol could be the intermediate compound for the production of [3H]alkyl- and [3H]alkenyl-phosphoethanolamine (plasmalogen) which were also metabolic products. FRTL5 cells were able to convert lyso-PAF to PAF especially when they were stimulated by ionophore A23187 in the presence of [3H]lyso-PAF and phenylmethylsulfonyl fluoride. The amount of PAF increased for the first 30 min and declined thereafter. PAF resting levels were found low in the same cells. Furthermore, PAF- acetylhydrolase activity was determined in cell homogenates. The presence of metabolic products such as alkyl-phosphatidylcholine, alkyl- and alkenyl- phosphatidylethanolamine and alkyl-glycerol, as well as, its mono- and diacyl derivatives, indicates that FRTL5 cells and probably other thyroid cells, are very active in metabolizing PAF and lyso-PAF and suggests the cooperation of the corresponding metabolic pathways in these cells.

CT Medical Descriptors:

*phospholipid metabolism

phospholipid synthesis

enzyme activity

cell labeling

bioassay

thyroid function

nonhuman

rat

controlled study

animal cell

article

priority journal

Drug Descriptors:

*thrombocyte activating factor: EC, endogenous compound

*1 o alkylglycero 3 phosphorylcholine: EC, endogenous compound

plasmalogen: EC, endogenous compound

RN (thrombocyte activating factor) 64176-80-3, 65154-06-5; (1 o alkylglycero 3 phosphorylcholine) 74430-89-0

AN 92032646 EMBASE

DN 1992032646

TI A specific, sensitive and high-capacity immunoassay for PAF.

AU Baldo B.A.; Smal M.A.; McCaskill A.C.

CS Kolling Inst./Medical Research, Royal North Shore Hospital, St. Leonards, NSW 2065, Australia

SO Lipids, (1991) Vol. 26, No. 12, pp. 1136-1139.

ISSN: 0024-4201 CODEN: LPDSAP

CY United States

DT Journal; Conference Article

FS 026 Immunology, Serology and Transplantation

029 Clinical Biochemistry

LA English

SL English

ED Entered STN: 920320

Last Updated on STN: 920320

AB A specific radioimmunoassay for **platelet-activating factor** (PAF) sensitive in the range 10-1000 pg (0.02-2 pmoles) has been developed. Detailed quantitative hapten inhibition studies showed specificity for the acetyl group at C-2 of PAF, a requirement for the ether linkage at C-1 and some tolerance for substituents on the choline nitrogen. No significant cross-reactivity was found with **phosphatidylcholine** and **lysophosphatidylcholine** or with lysoPAF.

CT Medical Descriptors:

*radioimmunoassay

*thrombocyte activation

conference paper

cross reaction

degranulation

intermethod comparison

intramuscular drug administration

nonhuman

priority journal

radioreceptor assay

sheep

standardization

structure activity relation

technique

thrombocyte aggregation

Drug Descriptors:

*thrombocyte activating factor: EC, endogenous compound

*thrombocyte activating factor derivative

RN (thrombocyte activating factor) 64176-80-3, 65154-06-5

AN 92032646 EMBASE

DN 1992032646

TI A specific, sensitive and high-capacity immunoassay for PAF.

AU Baldo B.A.; Smal M.A.; McCaskill A.C.

CS Kolling Inst./Medical Research, Royal North Shore Hospital, St. Leonards, NSW 2065, Australia

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CY United States

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029 Clinical Biochemistry

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Drug Descriptors:

*thrombocyte activating factor: EC, endogenous compound

*thrombocyte activating factor derivative

RN (thrombocyte activating factor) 64176-80-3, 65154-06-5

ANSWER 12 OF 16 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on
STN DUPLICATE 3

AN 1998:477824 BIOSIS

DN PREV199800477824

TI MgATP has different inhibitory effects on the use of 1-acyl-
lysophatidylcholine and lyso **platelet-activating factor** acceptors by neuronal nuclear
acetyltransferase activities.

AU Baker, R. Roy [Reprint author]; Chang, Huu-Yi

CS Div. Neurol., Dep. Med., Clin. Sci. Div., Rm. 6368, Med. Sci. Build.,
Univ. Toronto, Toronto, ON M5S 1A8, Canada

SO Biochimica et Biophysica Acta, (June 15, 1998) Vol. 1392, No. 2-3, pp.
351-360. print.

CODEN: BBACAO. ISSN: 0006-3002.

DT Article

LA English

ED Entered STN: 5 Nov 1998

Last Updated on STN: 5 Nov 1998

AB The inhibitory effects of MgATP on neuronal nuclear acetyltransferase activities were studied using lyso **platelet-activating factor** (lyso-PAF, 1-alkyl-sn-glycero-3-phosphocholine) and **lysophatidylcholine** (lyso-PC, 1-acyl-sn-glycero-3-phosphocholine). The nuclear (N1) acetylation of lyso-PC was more profoundly inhibited by MgATP. MgATP did not alter the apparent Km for acetyl-CoA in either acetylation reaction. The inhibitory effects of MgATP were not seen for other nucleotides or MgAMP-PCP. Kinase inhibitors such as staurosporine (1 μ M), chelerythrine, and R59022 (diglyceride kinase inhibitor 1) did not block the MgATP inhibition of either acetylation. However, the addition of phospholipids to the **assays** indicated a selective inhibitory effect for PIP (25-50 μ M) in the nuclear acetylation of lyso-PAF. When N1 was incubated with (γ -33P)ATP, phosphatidic acid and PIP were the principal radioactive lipid products. While the extent of MgATP inhibition of lyso-PAF acetylation was similar at different concentrations of lyso-PAF, increasing lyso-PC concentrations greatly decreased the MgATP inhibition seen in lyso-PC acetylations. Nuclear envelopes prepared in the presence of PMSF, and fraction N1 exposed to PMSF, did not show the inhibitory effect of MgATP on lyso-PC acetylation. PMSF (an inhibitor of certain phospholipase and lysophospholipase activities) did not reduce the MgATP inhibition of lyso-PAF acetylation. Arachidonoyl trifluoromethylketone, an inhibitor of cytosolic phospholipases A2 and of lysophospholipase activity associated with cPLA2, also blocked the inhibitory effect of MgATP on lyso-PC acetylation. Using radioactive lyso-PC substrate, fraction N1 produced labeled free fatty acid and **phosphatidylcholine**. In the presence of acetyl-CoA, the production of radioactive **phosphatidylcholine** increased almost 6-fold when MgATP was also included in these incubations. In the presence of MgATP and acetyl-CoA, PMSF reduced the levels of radioactive free fatty acid and **phosphatidylcholine** derived from lyso-PC, while Triacsin C, an inhibitor of acyl CoA synthetase, decreased **phosphatidylcholine** labeling. These findings suggest that MgATP inhibition of lyso-PC acetylation results from a loss of lyso-PC substrate that is largely mediated by nuclear lysophospholipase, acyl-CoA synthetase and lyso-PC acylation. Thus the neuronal nuclear production of Acyl PAF may be regulated by paths that compete for the lyso-PC substrate. In contrast, the acetylation of lyso-PAF is inhibited by PIP, a product of nuclear PI kinase reactions.

CC Enzymes - General and comparative studies: coenzymes 10802

Biochemistry methods - General 10050

Biophysics - Methods and techniques 10504

Biophysics - Molecular properties and macromolecules 10506

IT Major Concepts

Enzymology (Biochemistry and Molecular Biophysics); Methods and

Techniques
IT Chemicals & Biochemicals
 acetyltransferase: analysis, neuronal nuclear activity; arachidonoyl trifluoromethylketone: enzyme inhibitor; **lyso-platelet activating factor**: Cayman, enzyme substrate, Doosan-Serdary; magnesium-ATP: analysis, inhibitory effects; **phosphatidylcholine**: Doosan-Serdary; triacsin C: enzyme inhibitor; **1-acyl-lysophosphatidylcholine**
IT Methods & Equipment
 enzyme activity **assay**: activity **assays**, analytical method; lysophosphocholine metabolism **assay**: analysis/characterization techniques: CB, analytical method; neuronal nuclear fraction isolation: cell isolation method, isolation/purification techniques: CT
ORGN Classifier
 Leporidae 86040
Super Taxa
 Lagomorpha; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
 rabbit
Taxa Notes
 Animals, Chordates, Lagomorphs, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Vertebrates
RN 9012-30-0 (acetyltransferase)
52691-62-0Q (lyso-platelet activating factor)
74430-89-0Q (lyso-platelet activating factor)
108728-68-3Q (lyso-platelet activating factor)
1476-84-2 (magnesium-ATP)
76896-80-5 (triacsin C)

Techniques

IT Chemicals & Biochemicals

acetyltransferase: analysis, neuronal nuclear activity; arachidonoyl trifluoromethylketone: enzyme inhibitor; **lyso-platelet activating factor**: Cayman, enzyme substrate, Doosan-Serdary; magnesium-ATP: analysis, inhibitory effects; **phosphatidylcholine**: Doosan-Serdary; triacsin C: enzyme inhibitor; **1-acyl-lysophosphatidylcholine**

IT Methods & Equipment

enzyme activity **assay**: activity **assays**, analytical method; lysophosphocholine metabolism **assay**: analysis/characterization techniques: CB, analytical method; neuronal nuclear fraction isolation: cell isolation method, isolation/purification techniques: CT

ORGN Classifier

Leporidae 86040

Super Taxa

Lagomorpha; Mammalia; Vertebrata; Chordata; Animalia

Organism Name

rabbit

Taxa Notes

Animals, Chordates, Lagomorphs, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Vertebrates

RN 9012-30-0 (acetyltransferase)

52691-62-0Q (**lyso-platelet activating factor**)

74430-89-0Q (**lyso-platelet activating factor**)

108728-68-3Q (**lyso-platelet activating factor**)

1476-84-2 (magnesium-ATP)

76896-80-5 (triacsin C)

on STN

AN 1998195672 EMBASE

TI Metabolic fate of **platelet-activating factor**
(PAF, 1-O-alkyl-2-acetyl- sn-glycero-3-phosphocholine) and
lyso-PAF (1-O-alkyl-2-lyso-sn-glycero-3- phosphocholine) in
FRTL5 cells.

AU Botitsi E.; Mavri-Vavayanni M.; Siafaka-Kapadai A.

CS A. Siafaka-Kapadai, Dept. of Chemistry (Biochemistry), University of
Athens, Zografou, 15771 Athens, Greece

SO Journal of Lipid Research, (1998) Vol. 39, No. 6, pp. 1295-1304.

Refs: 47

ISSN: 0022-2275 CODEN: JLPRAW

CY United States

DT Journal; Article

FS 029 Clinical Biochemistry

LA English

SL English

ED Entered STN: 19980716

Last Updated on STN: 19980716

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(PAF, 1-O-alkyl-2-acetyl- sn-glycero-3-phosphocholine) and
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investigated in FRTL5 cells, a normal rat thyroid cell line. FRTL5 cells
incorporated [3H]PAF and deacetylated this compound to the corresponding
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alkyl-acyl-**phosphocholine** indicating that this acylation process
was particularly active in these cells. Among metabolic products of both
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(plasmalogen) which were also metabolic products. FRTL5 cells were able
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ionophore A23187 in the presence of [3H]lyso-PAF and phenylmethylsulfonyl
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thereafter. PAF resting levels were found low in the same cells.
Furthermore, PAF- acetylhydrolase activity was determined in cell
homogenates. The presence of metabolic products such as alkyl-
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and alkyl-glycerol, as well as, its mono- and diacyl derivatives,
indicates that FRTL5 cells and probably other thyroid cells, are very
active in metabolizing PAF and lyso-PAF and suggests the cooperation of
the corresponding metabolic pathways in these cells.

CT Medical Descriptors:

- *phospholipid metabolism
- phospholipid synthesis
- enzyme activity
- cell labeling
- bioassay
- thyroid function
- nonhuman
- rat
- controlled study
- animal cell
- article
- priority journal

Drug Descriptors:

- *thrombocyte activating factor: EC, endogenous compound
- *1 o alkylglycero 3 phosphorylcholine: EC, endogenous compound
- plasmalogen: EC, endogenous compound

RN (thrombocyte activating factor) 64176-80-3, 65154-06-5; (1 o alkylglycero
3 phosphorylcholine) 74430-89-0

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ANSWER 12 OF 16 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on
STN DUPLICATE 3

AN 1998:477824 BIOSIS

DN PREV199800477824

TI MgATP has different inhibitory effects on the use of 1-acyl-
lysophatidylcholine and lyso platelet-
activating factor acceptors by neuronal nuclear
acetyltransferase activities.

AU Baker, R. Roy [Reprint author]; Chang, Huu-Yi

CS Div. Neurol., Dep. Med., Clin. Sci. Div., Rm. 6368, Med. Sci. Build.,
Univ. Toronto, Toronto, ON M5S 1A8, Canada

SO Biochimica et Biophysica Acta, (June 15, 1998) Vol. 1392, No. 2-3, pp.
351-360. print.

CODEN: BBACAO. ISSN: 0006-3002.

DT Article

LA English

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AB The inhibitory effects of MgATP on neuronal nuclear acetyltransferase activities were studied using lyso platelet-activating factor (lyso-PAF, 1-alkyl-sn-glycero-3-phosphocholine) and lysophatidylcholine (lyso-PC, 1-acyl-sn-glycero-3-phosphocholine). The nuclear (N1) acetylation of lyso-PC was more profoundly inhibited by MgATP. MgATP did not alter the apparent Km for acetyl-CoA in either acetylation reaction. The inhibitory effects of MgATP were not seen for other nucleotides or MgAMP-PCP. Kinase inhibitors such as staurosporine (1 μ M), chelerythrine, and R59022 (diglyceride kinase inhibitor 1) did not block the MgATP inhibition of either acetylation. However, the addition of phospholipids to the assays indicated a selective inhibitory effect for PIP (25-50 μ M) in the nuclear acetylation of lyso-PAF. When N1 was incubated with (γ -33P)ATP, phosphatidic acid and PIP were the principal radioactive lipid products. While the extent of MgATP inhibition of lyso-PAF acetylation was similar at different concentrations of lyso-PAF, increasing lyso-PC concentrations greatly decreased the MgATP inhibition seen in lyso-PC acetylations. Nuclear envelopes prepared in the presence of PMSF, and fraction N1 exposed to PMSF, did not show the inhibitory effect of MgATP on lyso-PC acetylation. PMSF (an inhibitor of certain phospholipase and lysophospholipase activities) did not reduce the MgATP inhibition of lyso-PAF acetylation. Arachidonoyl trifluoromethylketone, an inhibitor of cytosolic phospholipases A2 and of lysophospholipase activity associated with cPLA2, also blocked the inhibitory effect of MgATP on lyso-PC acetylation. Using radioactive lyso-PC substrate, fraction N1 produced labeled free fatty acid and phosphatidylcholine. In the presence of acetyl-CoA, the production of radioactive phosphatidylcholine increased almost 6-fold when MgATP was also included in these incubations. In the presence of MgATP and acetyl-CoA, PMSF reduced the levels of radioactive free fatty acid and phosphatidylcholine derived from lyso-PC, while Triacsin C, an inhibitor of acyl CoA synthetase, decreased phosphatidylcholine labeling. These findings suggest that MgATP inhibition of lyso-PC acetylation results from a loss of lyso-PC substrate that is largely mediated by nuclear lysophospholipase, acyl-CoA synthetase and lyso-PC acylation. Thus the neuronal nuclear production of Acyl PAF may be regulated by paths that compete for the lyso-PC substrate. In contrast, the acetylation of lyso-PAF is inhibited by PIP, a product of nuclear PI kinase reactions.

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IT Major Concepts

Enzymology (Biochemistry and Molecular Biophysics); Methods and